PB 198 581

A I R B O R N E A S B E S T O S

A Report Prepared by the

Committee on Biologic Effects of Atmospheric

Pollutants

of the

Division of Medical Sciences, National Research

Council

National Academy of Sciences

National Academy of Engineering Washington, D.C. 1971

Reproduced by
NATIONAL TECHNICAL
INFORMATION SERVICE
Springfield, Va. 22151

61

Committee on Biologic Effects of Atmospheric Pollutants, Division of Medical Sciences, National Research Council:

Dr. Arthur B. DuBois, Department of Physiology, School of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania, Chairman

Mr. Vinton W. Bacon, College of Applied Science and Engineering, University of Wisconsin, Milwaukee, Wisconsin

Dr. Anna M. Baetjer, Department of Environmental Medicine, School of Hygiene and Public Health, The Johns Hopkins University, Baltimore, Maryland

Dr. W. Clark Cooper, School of Public Health, University of California, Berkeley, California

Dr. Morton Corn, Graduate School of Public Health, University of Pittsburgh, Pittsburgh, Pennsylvania

Dr. Bertram D. Dinman, School of Public Health, University of Michigan, Ann Arbor, Michigan

Dr. Leon Golberg, Institute of Experimental Pathology and Toxicology, Albany Medical College, Albany, New York

Dr. Paul B. Hammond, Department of Physiology and Pharmacology, College of Veterinary Medicine, University of Minnesota, St. Paul, Minnesota

Dr. Samuel P. Hicks, Department of Pathology, University of Michigan Medical Center, Ann Arbor, Michigan

Dr. Victor G. Laties, Department of Radiation Biology and Biophysics, University of Rochester Medical Center, Rochester, New York

Dr. Abraham M. Lilienfeld, Department of Chronic Diseases, School of Hygiene and Public Health, The Johns Hopkins University, Baltimore, Maryland

Committee on Biologic Effects of Atmospheric Pollutants, Division of Medical Sciences, National Research Council (cont'd.):

Dr. Paul Meier, Biomedical Computation Facilities, University of Chicago, Chicago, Illinois

Dr. James N. Pitts, Jr., Department of Chemistry, University of California, Riverside, California

Dr. Gordon J. Stopps, Haskell Laboratory, E. I. duPont de Nemours and Company, Newark, Delaware

Dr. O. Clifton Taylor, Department of Horticulture, University of California, Riverside, California

Dr. Jaroslav J. Vostal, Department of Pharmacology and Toxicology, University of Rochester Medical Center, Rochester, New York

Executive Director. T. D. Boaz, Jr., M.D.

Panel on Asbestos:

Dr. W. Clark Cooper, School of Public Health, University of California, Berkeley, California, Chairman

Dr. Lewis J. Cralley, Bureau of Occupational Health and Safety, Division of Epidemiology and Special Services, U. S. Public Health Service, Cincinnati, Ohio

Dr. Benjamin G. Ferris, Jr., Department of Physiology, Harvard School of Public Health, Boston, Massachusetts

Dr. Paul Gross, Industrial Hygiene Foundation, Inc., Pittsburgh Pennsylvania

Mr. Duncan A. Holaday, Occupational Health Field Station, Salt Lake City, Utah

Dr. Irving J. Selikoff, Environmental Sciences Laboratory, Mount Sinai Hospital, New York, New York

Dr. George W. Wright, Medical Research Department, St. Luke's Hospital, Cleveland, Ohio

Dr. Samuel P. Hicks, Department of Pathology, University of Michigan Medical Center, Ann Arbor, Michigan, Associate Editor

Dr. T. D. Boaz, Jr., Division of Medical Sciences, National Research Council, Washington, D.C., Staff Officer

CONTENTS

		Page
Chapter 1	Pathogenicity of Asbestos.....	1
Chapter 2	Evidence of Human Nonoccupational Exposures.....	11
Chapter 3	Estimation of Risk in Nonoccupational Exposures.....	16
Chapter 4	Sources of Asbestos Fibers in Ambient Air.....	21
Chapter 5	Principles of Control.....	26
Chapter 6	Research Needs.....	30
Chapter 7	Conclusions and Recommendations.....	32
	References.....	34

PREFACE

The naturally occurring fibrous silicates classified as "asbestos" have become almost indispensable in modern technology.[67,125,142] The world's annual production has grown from a few thousand tons in 1900 to over 3 million tons in 1968.[121] Annual consumption in the United States averaged nearly 800,000 tons during the period 1965-1969.[121] The potential of asbestos as a hazard to health has been the subject of a number of reviews in recent years.[22,49,60,134,137,146,148,168]

Although it has been known for a half-century that persons who inhaled large amounts of asbestos dust in the course of their work sometimes developed disabling or fatal fibrosis of the lungs, it has been only within the last three decades that other serious effects, such as cancer, have been associated with occupational exposures. Recently, the likelihood of exposure of the public at large to asbestos has been recognized and has led to a demand for more rigorous control of asbestos emissions into the atmosphere.

"Asbestos" is a generic term for a number of hydrated silicates that, when crushed or processed, separate into flexible fibers made up of fibrils.[46] Although there are many asbestos minerals, only six are of commercial importance: chrysotile, a tubular serpentine mineral, accounts for 95% of the world's production; the others, all amphiboles, are amosite, crocidolite, anthophyllite, tremolite, and actinolite. The asbestos minerals differ in their metallic elemental content, range of fiber diameters, flexibility or harshness, tensile strength, surface properties, and other

attributes that determine their industrial uses and may affect their respirability, deposition, retention, translocation, and biologic reactivity.

This report (1) summarizes the major evidence of the pathogenicity of asbestos in man and animals, (2) summarizes the evidence of human nonoccupational exposure to asbestos, (3) evaluates the evidence of a health risk associated with various degrees and types of exposure, (4) identifies sources of environmental contamination by asbestos, and (5) offers recommendations concerning the need for and feasibility of control measures.

W. Clark Cooper
Chairman

CHAPTER 1

PATHOGENICITY OF ASBESTOS

The effects of fibers in biologic systems may result not only from the properties of the fibers themselves, but also from contamination with inorganic or organic substances that occur naturally or are added during mining, milling, processing, shipping, or use. Contaminants acquired from the atmosphere or in the respiratory tract may be carried on the surface of fibers. Fibers may act as cofactors; conversely, their action may be modified by other cofactors.

PATHOGENICITY IN MAN

The proven or suspected effects of asbestos minerals on human health include nonmalignant changes, such as pulmonary and pleural fibrosis, and several types of malignancy, notably of the lung, pleura, and peritoneum. Nearly all the positive evidence of an association between asbestos and human disease has come from occupational groups. With few exceptions, these have consisted of workers engaged in the mining and milling of asbestos, the manufacture of asbestos-containing products (such as textiles and construction materials), and the application and removal of asbestos-containing insulating materials.

Asbestosis

Asbestosis, or asbestotic pneumoconiosis, was the first clearly demonstrated adverse effect of asbestos in man. It is characterized by a pattern of roentgenographic changes in the lung consistent with diffuse interstitial fibrosis of variable degree and at times with fibrosis and calcification of the pleura; clinical changes that include fine rales,

finger clubbing, and shortness of breath, each of which may be absent in an individual case; and physiologic changes consistent with a restrictive lung disorder.

The first published mention of a case, in a man who had worked for 10 years in the carding room of an asbestos factory, was by H. Montague Murray in 1907.[111] Cooke reported a second case in 1924[20] and in 1927 provided a more detailed description,[21] in which the term "asbestosis" was first used. In 1930, Merewether[105] reviewed the salient features of the disease and the environmental exposures of workers, including data derived from an epidemiologic study reported in more detail by Merewether and Price.[106] This led to the promulgation of regulations for environmental and medical control in the United Kingdom, which became effective in 1932.[7] Cases were first reported in the United States in 1930,[110,141] and guidelines for acceptable dust concentrations were proposed by Dreessen et al. in 1938.[32]

Industrial experience indicates that pulmonary fibrosis sufficient to interfere with respiratory or cardiovascular function can be prevented by reducing asbestos dust concentrations to levels that are still far above any likely to be encountered in community air.

Pleural Calcification

Calcified pleural plaques occur frequently in workers exposed to asbestos.[5,6,43,103,108,130] When multiple or bilateral, they are regarded by some as almost diagnostic of asbestos-related disease.[43,71] In asbestos workers, calcified plaques rarely appear until 20 years after first exposure and do not necessarily correlate with parenchymal fibrosis.[91]

Calcification appears to differ in frequency in different occupationally exposed groups, but studies are inadequate to verify or explain such differences.

Bronchogenic Carcinoma

No features of bronchogenic carcinoma associated with asbestos are pathognomonic. Primary sites are more often in the lower lobes, in contrast with the usually higher frequency of upper lobe tumors. Peripheral primary sites are common in asbestos-related lung cancer. All cell types are represented in most series.

The first suggestion that asbestos might be causally related to cancer of the lung was made in 1935 by Lynch and Smith,[91] who described squamous cell carcinoma in a South Carolina textile worker with asbestosis. Despite other isolated reports, an association was not firmly supported by epidemiologic evidence until 1947, when Merewether, Chief Inspector of Factories in the United Kingdom, reported 31 instances of cancer of the lung in 235 persons known by his department to have died with asbestosis between 1924 and 1946.[104] That constituted an incidence of 13.2%, compared with 1.32% (91/6884) in persons certified as having had silicosis during the same period. Gloyne in 1951[50] reported on the pathologic findings in 1205 lungs from workers whose cases were being evaluated for pneumoconiosis. In 132 asbestos workers, he found 121 with asbestosis, of whom 17, or 14.0% had cancer of the lung, compared with 55 (6.9%) of 796 persons who had silicosis. The study was done, however, in a hospital to which suspected tumor patients would have been referred; thus, there may have been an overestimate of risk. Doll in 1955,[31] after analyzing the causes

of death among 105 men who had worked for at least 20 years in areas of asbestos textile plants defined as dusty, concluded that the 18 cases of lung cancer that occurred indicated a risk about 10 times that in the general male population.

Other studies[11,13,15,18,23,24,33,34,38-40,59,70,74-76,80-82,87,93,94,98,101,112,131,149,154,155,161,169] have confirmed an association between occupational exposure to asbestos and a highter-than-expected incidence of bronchogenic cancer. Some studies[13] have demonstrated differences in the degree of risk among different occupationally exposed groups, probably related to dose, as well as to other factors.

Mesothelial Tumors

Primary malignant tumors of the pleura and peritoneum have been regarded as exceedingly rare by most pathologists; until recent years, some even questioned their existence. There are still differences of opinion as to diagnostic criteria.[18,70,98,149] Therefore, statistics on prevalence or incidence in one geographic area cannot be safely compared with those from another. Adherence to strict diagnostic criteria, including an autopsy complete enough to rule out other primary tumors that could metastasize to or involve serosal surfaces, is a difficult constraint on a retrospective series.

It was after 1960 that serious consideration was first given to asbestos as an etiologic factor in mesothelial malignancies. In that year, Wagner et al.[161] reported 33 cases of pleural mesothelioma in a part of South Africa important for crocidolite mining. For all but two of the patients, the authors discovered likely asbestos contacts two decades or

more earlier. However, only 17 of these had had occupational exposure. The remainder had lived near mines or had had household contacts. Although mesothelioma had previously been attributed by some to asbestos exposures,[86,164,165,169] no evidence of a strong association had been developed.

Additional information supporting a relationship between asbestos and malignant mesothelioma has accumulated since 1960.[9,12,36,37,41,42,59,75,80,88,93,95,99,100,102,113-116,132,134,136] An outstanding feature has been the long period, commonly over 30 years, between the first exposure to asbestos and the appearance of a tumor, as emphasized in reports by Wagner[158] and Selikoff et al.[134]

Other Neoplasia

Associations between asbestos exposures and malignancies of the gastrointestinal tract and of other sites have been reported, but the data are still inconclusive.[52,59,77,94,134]

Mutagenic Effects

There is no evidence that asbestos is associated with mutagenesis.

PATHOGENICITY IN LOWER ANIMALS

A comprehensive review of information derived from experimental work in animals entails consideration not only of the variables related to the type and dimensions of the asbestos fibers and of adsorbed or concurrently administered contaminants, but also of the species and strain of animal, the route of administration, and the time and dosage factors. There are at present no satisfactory experimental models to duplicate prolonged inhalation of asbestos by man, but many isolated segments of the problems have been elucidated. The studies are best divided into those dealing with asbestosis and those dealing with neoplasia.

Experimental Asbestosis

Asbestotic pulmonary fibrosis has been produced experimentally in various species of animals, including rats,[45,58,156,157] guinea pigs,[29,45,69,157] hamsters,[55] rabbits,[157] and monkeys.[157] In many of the studies, the disease resembled early asbestotic development in man--e.g., it was multifocal. Diffuse fibrosis has also been produced,[157] but to do so it was necessary to use very high concentrations of asbestos dust and long periods of exposure or observation after exposure. (In contrast, experimental silicosis can be produced with lower cumulative exposure.) In the course of the investigations, it has been asked whether the fibrogenicity of asbestos dust is mostly confined to fibers longer than 5 μm;[45,156] the question is still unanswered.

Experimental Neoplasia

Lung cancer from chrysotile dust has been produced experimentally in rats[58] and in mouse lung implants.[140] Other investigators who used different methods for introducing the dust[143] did not find lung cancer in the animals they studied. That some asbestos dust has an increased content of trace metals--particularly nickel, chromium, and cobalt--may explain these differing experiences. Rats whose lung clearance had been artificially impaired had twice the lung cancer rate of animals with normal clearance.[58]

Cancer of the pleural surface (mesothelioma) has been reported in rats and hamsters that received intrapleural injections of the three most common types of asbestos.[139,159,160] The amounts of asbestos dust introduced into the thoracic cavity were very large, and translation of results to human inhalation of asbestos is uncertain.

The studies of Roe et al.,[124] which involved pleural and peritoneal mesotheliomas of mice after subcutaneous injections of crocidolite, amosite, or chrysotile, are of particular interest because they yielded evidence of migrations of fibers.

Naturally Occurring Effects in Lower Animals

There is no evidence that effects on domestic or wild animals are important as criteria for controlling asbestos emissions. Schuster has described pulmonary asbestosis (without the development of asbestos bodies) in a dog that lived for nearly 10 years as a ratcatcher in a London asbestos factory.[128] Webster has demonstrated fibrosis in donkeys, baboons, and wild rodents in South Africa,[163] and Kiviluoto has described the finding of anthophyllite asbestos in the lungs of a cow in an anthophyllite-producing area of Finland.[79]

STUDIES IN VITRO

There have been limited studies of the effects of asbestos in biologic systems in vitro. For example, MacNab and Harington[92] demonstrated in 1967 that asbestos would hemolyze sheep erythrocytes. This has been confirmed by others.[127,129] Although chrysotile is markedly hemolytic, amosite, crocidolite, and anthophyllite have little or no activity under similar conditions.

Parazzi et al.[117] demonstrated in 1968 that both crocidolite and chrysotile were cytotoxic for guinea pig macrophages in culture. The activity of the former was greater; the cytotoxicity of neither was innibited by

polyvinylpyridine N-oxide, a macromolecular chemical that is effective in preventing the cytotoxic damage caused by crystalline silica in experimental conditions.

Although the foregoing types of study have no known relationship to fibrogenic or carcinogenic effects *in vivo*, they provide systems that may prove useful in determining mechanisms of action and approaches to prophylactic or therapeutic measures.

INFLUENCE OF MAJOR VARIABLES ON PATHOGENICITY

When considering the importance of type of asbestos, fiber size, and cofactors on biologic effects, it is necessary to emphasize that a given attribute may influence in differing ways the respirability, deposition, retention, clearance, translocation, and biologic reactivity. Although some *in vitro* and laboratory studies yield different responses to different types of asbestos, the results do not justify drawing firm conclusions as to the relative pathogenicity of the different types. Nor do epidemiologic studies conclusively support such differences. All the commercially used forms of asbestos can produce asbestosis. In only relatively few studies has the incidence of malignancies been determined in groups with exposures to a single asbestos type. Where there are data that suggest a lower risk, as in the chrysotile-producing areas of Canada[13,100] and Italy,[154] there are possible explanations for the difference other than asbestos type. The high incidence of mesothelial tumors in the North Western Cape area of South Africa has led to the suggestion that crocidolite is unusually hazardous, but mesotheliomas have been rare in the Transvaal, where crocidolite is also produced.[49,138] Although Selikoff *et al.*[132] found

many mesotheliomas in insulation workers whose exposures had been largely to chrysotile and amosite, Sluis-Cremer[138] and Webster[162] have not found the incidence of mesothelioma high in areas where amosite was mined and milled, and McDonald[101] did not report an excess in the chrysotile mining and milling areas of Canada.

All epidemiologic studies that appear to indicate differences in pathogenicity among types of asbestos are flawed by their lack of quantitative data on cumulative exposures, fiber characteristics, and the presence of cofactors. The different types, therefore, cannot be graded as to relative risk with respect to either asbestosis or neoplasia.

Fiber size is critically important in determining respirability, deposition, retention, and clearance from the pulmonary tract and is probably an important determinant of the site and nature of biologic action. Little is known about the movement of fibers within the body, including their potential for entry through the gastrointestinal tract. The aerodynamic properties of fibers depend largely on their diameter; fibers below 3.5 μm in diameter are regarded as being in the respirable range.[152] Fiber length affects deposition, longer fibers apparently having greater fibrogenic effects.[44,156] Until recently, most work and speculation have involved only fibers detectable by the optical or light microscope (LM), approximately 0.5 μm in diameter and larger. The application of electron microscopic (EM) techniques[134] has enlarged our horizons as to the variables that must be considered. Tissues and air samples may contain many EM-sized fibrils for every LM-sized fiber that can be demonstrated.[84,134] There is, however, no body of knowledge that permits the assigning of relative risk factors to fibers in the EM

range, compared with fibers in the LM range. It is possible that the relative risks associated with fibers of different sizes are different for nonmalignant and malignant changes. The evidence that bundles of fibrils may be broken down within the body to individual fibrils is important.[147]

A number of investigators have postulated that a probable role of asbestos fibers in producing disease is to carry toxic or oncogenic substances to vulnerable sites. Studies bearing on this have included analysis of various types of asbestos to determine the contaminants present, with special emphasis on metals[26,27] and polycyclic hydrocarbons;[48,61-64] studies of the elution of contaminants in biologic materials;[26] and the concurrent exposure of animals to asbestos and to other materials.[109] The present consensus is that contaminants are present, but a special pathogenetic role is still speculative.

The work of Selikoff et al.[135] strongly suggests a synergism of cigarette smoking and asbestos exposure in the increased risk of lung cancer in insulation workers. It is not known whether this is because of reduced clearance of asbestos, transportation of cigarette-smoke carcinogens by asbestos fibers, or the promotion by one factor of cancer initiated by another.

CHAPTER 2

EVIDENCE OF HUMAN NONOCCUPATIONAL EXPOSURES

Direct and indirect evidence that persons other than those working directly with asbestos minerals are being exposed to asbestos is of several types. For example, asbestos fibers can be demonstrated in the lungs of persons not occupationally exposed. In a few geographic areas, pathologic changes regarded as representing a reaction to asbestos (e.g., pleural calcification) have been found in populations with no history of occupational exposure. Asbestos fibers have been demonstrated in ambient air.

FIBERS IN LUNG TISSUE

Structures that appear to be fibers coated with a pigmented material were described in lung tissue as early as 1907 by Marchand.[96] These structures were actually fibers coated with hemosiderin. In 1929, Cooke[19] described such "curious bodies" in association with pulmonary fibrosis. Stewart and Haddow[144] referred to them as "asbestosis bodies." Because those who work with asbestos exhibit them a few months after starting work, it was recognized that they were evidence of exposure, but not of asbestosis. The term "asbestos body" came to be the preferred designation.

As long as the coated fibers were found in persons known to have been occupationally exposed to asbestos, the identity of the central fiber was seldom questioned, although from time to time similar objects were found in persons with no known exposure to asbestos. Meurman[107] in 1966 summarized 19 reports published between 1932 and 1962 in which these objects were associated with exposure to graphite, coal, hornblende, rutile, diatomaceous earth, carborundum, and talc (in which case tremolite asbestos might have been involved). The demonstration by Gross[54,56] that other fibers may

produce such bodies in experimental animals indicates that they result from a nonspecific reaction to any sparingly soluble fibrous foreign body, as had first been suggested by Cooke in 1929.[19] There is thus ample justification for abandoning "asbestos body" as a generic term; Gough[51] in 1965 suggested the term "mineral fiber-body," and Gross[53] in 1966 recommended "ferruginous body."

Thomson et al.,[151] in 1963, were the first to show that these coated fibers were present in a high proportion of lungs obtained by routine autopsy. They found that 26.4% of lung smears in 500 consecutive autopsies in Cape Town showed what were called "asbestos bodies." Reports from many other areas have confirmed a high prevalence in lungs obtained in similar autopsy series.[3,8,17,30,47,107,118,123,150] Utidjian et al.[153] inferred that, if a sufficient volume of lung tissue were examined in each case, nearly all persons would be found to have such bodies; their study of 100 lungs in Pittsburgh confirmed their suggestion.

Identification of the core fibers has proved to be a formidable technical task.[57,84,119] Without fiber-by-fiber analysis, all that can be said is that coated fibers resembling those in asbestos workers are present in most persons in our urban centers. Stripping the coating and analyzing the cores by various techniques can sometimes demonstrate that the cores are asbestos, but the process is tedious and often inconclusive.

Attention is now being directed toward study, not of the ferruginous bodies alone, but of the total fiber content of the lungs, whether such fibers are coated or uncoated. In a study of 3,000 consecutive autopsies in New York City, Langer et al.[84] have found thin, uncoated, optically

visible fibers in two-thirds of the 1,449 lung specimens in which coated fibers were demonstrated and in one-fourth of those in which uncoated fibers were demonstrated. Twenty-eight consecutive samples of lung tissue from the same series examined by electron microscopy were found to contain EM-sized chrysotile fibers.[85] Pooley et al.[119] have reported similar findings.

Evidence is therefore strong that most human lungs harbor thousands or millions of fibers. Some of these are chrysotile asbestos, and other types of asbestos minerals are probably there also. In most persons not occupationally exposed to asbestos, the numbers of fibers are relatively small, compared with the numbers found in the occupationally exposed.[133] The systematic application of quantitative techniques, measuring both coated and uncoated fibers, is needed to define a gradient of accumulated fibers for correlation with incidence of disease, on the one hand, and history of environmental exposure, on the other.

Although there appears no doubt that asbestos fibers are present in many human lungs, there are sources of airborne fibers other than asbestos.[28,57] Some are probably derived from the burning of leaves and plant products, such as paper, wood, and coal. Man-made (mostly vitreous) fibers have also been identified in the sediment isolated from human lungs. Talc, often used generously as a dusting powder, may contain a significant amount of tremolite asbestos fibers.

Information is sparse concerning possible increase of fibers in lungs with increasing use of asbestos and concerning the existence of significant differences between urban and rural populations. Selikoff and Hammond[133] compared lung tissues obtained in 1934 and 1967 and found no significant

increase in the proportion containing ferruginous bodies. This suggested that, despite increasing use of asbestos in New York City between 1934 and 1967, fibers of a type producing ferruginous bodies had not been increasing at a corresponding rate. However, Chang-Hyun Um[73] reports an increase over each decade in asbestos bodies in samples of lungs from persons who died in London in 1936, 1946, 1956, 1966.

PLEURAL CALCIFICATION IN THE GENERAL POPULATION

Meurman[108] in 1968 reviewed critically the literature related to pleural calcification and asbestos exposure. A number of studies strongly suggested an association between pleural calcification and nonoccupational exposures to asbestos. For example, Kiviluoto[79] in 1960 reported calcifications in 9% of the adult population detected during mass roentgenographic surveys in a Finnish commune in which there was an asbestos mine and mill; the frequency was low for the remainder of the Finnish population. Raunio[120] enlarged on these observations in 1966, reporting that, of 633,201 chest films taken in Finland between 1960 and 1965, 1516 showed pleural calcifications; 1232 of the latter were among 43,483 films taken in 10 communes in which there were anthophyllite mines. Rock and soil in such areas also contain much asbestos, so that the demonstration that airborne anthophyllite could be demonstrated over 25 km from the mines is not necessarily relevant. Anspach[4] reported that, of 244 subjects with pleural calcification found in a chest roentgenographic survey in Dresden, 177 had either worked in or lived near an asbestos factory. Zolov et al.[170] described a 5.1% prevalence of pleural calcification in a rural population in Bulgaria and suggested that the most likely cause was asbestos in the soil. However, Hromek,[72] Marsová,[97] and Rous and

Studený,[126] reporting on a high prevalence of pleural plaques in a rural district of Czechoslovakia, have been unable to demonstrate a source of asbestos exposure. The consensus at present is that calcification alone may not invariably be considered an index of asbestos exposure in the general population, although it may prompt a search for an environmental source of asbestos.

MEASUREMENT OF AIRBORNE ASBESTOS

A more direct method of obtaining evidence on the likelihood of exposure of the general population would be the sampling of air to determine the presence and amount of respirable asbestos fibers. There are, however, many uncertainties as to the best methods of sampling, identifying, and quantitating airborne asbestos and interpreting data so obtained.[78,90,122,137] Limited information has been derived from measuring fibers on sampling sites, such as that by Laamanen et al.,[83] who showed asbestos fallout diminishing rapidly beyond 1 km from an anthophyllite quarry, but still detectable at 27 km. Counts of asbestos fibers collected on membrane filters by high-volume air sampling and estimated by light microscopic techniques similar to those used in industrial hygiene have shown small numbers of fibers in a few urban sites.[10,16] Such results, although showing numbers of fibers detectable by the light microscope that were low by occupational health experience, have been too few and variable to be used with confidence. Alternative methods that are currently under development, including estimations of the number and mass of fibers in the LM and the EM size ranges, have shown measurable concentrations of asbestos in many samples of ambient air.[1,66,137] Such environmental measurements are in their earliest stages and provide few clues to the extent or significance of the risk from this type of exposure to asbestos or to other mineral fibers.

CHAPTER 3

ESTIMATION OF RISK IN NONOCCUPATIONAL EXPOSURES

Industrial experience has shown that prolonged inhalation of asbestos can increase the risk of neoplastic disease. Examination of lung tissue has made it apparent that a much larger proportion of the general public has inhaled and retained asbestos fibers than had formerly been realized; in fact, most urban dwellers have some such fibers in their lungs. Can these facts be related? Does the general public--as well as persons working near occupational sources, living in the households of asbestos workers, living in the neighborhoods of asbestos plants, or having occasional random exposures--have a detectably increased risk of malignancy or other disease because of airborne asbestos? The limited information we have to answer these questions comes either from direct epidemiologic studies of groups with various levels of nonoccupational exposure or by extrapolation from the experience of industrial populations with direct or indirect asbestos exposures.

EPIDEMIOLOGIC STUDIES RELATED TO NONOCCUPATIONAL EXPOSURES

Two general indices of asbestos exposure are available for use in direct epidemiologic studies of groups not known to be occupationally exposed to asbestos. The first is based on knowledge of each member's place of work and place of residence; because of the long latent periods of asbestos-related disease, this knowledge must cover each person's whole lifetime. The second is a quantitative estimate of each member's lung content of asbestos fibers. There are few such direct epidemiologic studies, and they are inadequate to answer the questions at issue.

The only studies that appear to implicate asbestos in the development of malignancies in persons not occupationally exposed are those involving diffuse mesothelioma, a tumor that is uncommon and that has been the subject of special attention in recent years. Many of the mesotheliomas reported by Wagner et al.[161] in South Africa were attributed to household and neighborhood exposures in a crocidolite-producing area. Although non-occupational, these exposures have been described as substantial.[168] Newhouse[114] studied 76 patients with mesothelioma diagnosed in London Hospital from 1917 to 1964. Of these 31 (40.8%) had occupational exposures to asbestos, 9 (11.8%) had a relative who worked with asbestos, 11 (14.5%) had neither of those backgrounds but had lived within a half-mile of an asbestos factory, and 25 (32.9%) had no known contacts. Corresponding percentages for a group of matched control subjects (patients in the same hospital for other diseases) were 10.5%, 1.3%, 6.6%, and 81.6%. Stumphius and Meyer[145] reported no mesotheliomas in the community near the shipyard in Flushing (Holland), although 17 of 21 mesotheliomas reported in the province of Zeeland in 1964-1967 had been in workers in that yard. Raunio[120] found no excess of pulmonary, pleural, or peritoneal malignancies in the areas of Finland where pleural calcifications attributed to anthophyllite were present in 6%-9% of routine chest survey films. In a series of 17 mesotheliomas collected by Borow et al.,[12] all but two were in persons who had worked in an asbestos mill, although the autopsy series from which the cases were drawn came from an area that included inhabitants of the mill's environs. Lieben and Pistawka[87] found that of 42 persons with mesotheliomas reported in Pennsylvania, 10 had worked in asbestos plants, 8 lived or worked close to an asbestos industry, and 3 were members of families that

included asbestos workers; in 11, no history of exposure could be obtained, and the remaining 10 had questionable random exposures. McDonald et al.[100] collected information on 165 fatal malignant mesotheliomas known to pathologists in Canada between 1959 and 1968. They confirmed an association with occupational exposure to asbestos but concluded that the excess was in the manufacture and industrial application of asbestos, rather than in mining or milling. It is apparent that no quantitative conclusions were possible from these studies, which present serious methodologic problems to the epidemiologist. They suggest a risk in household contacts and in residence in the immediate neighborhood of asbestos plants. There appear to be different levels of risk in different types of occupational exposures, and some of these may be reflected in corresponding household and neighborhood experience.

In no analysis of causes of death in a large population has there been quantitative estimation of the lung content of ferruginous bodies and bare asbestos fibers, to determine whether a detectable gradient of disease can be correlated with asbestos content. The series so far studied have been too small, and methods have been too variable, to permit any conclusions as to the importance of small numbers of fibers in the lung.

EXTRAPOLATION FROM OCCUPATIONAL EXPERIENCE

Another source of evidence of the relative risks associated with inhaling moderate or small numbers of asbestos fibers is the experience of persons who have had occupational exposures below those known to be definitely hazardous. The maximal airborne fiber concentrations recommended for prevention of asbestosis are much higher than any likely to be encountered in

nonoccupational situations. For example, one recommended standard would limit the average concentration of airborne chrysotile to 2000 fibers per liter as determined by light-field count.[14] Another that has been proposed would limit average concentrations of fibers to 5000 fibers per liter.[2] Occupation-related asbestosis can be effectively controlled with airborne fiber concentrations much higher than are likely to be encountered in non-occupational situations. It is important to determine whether workers whose exposures have been reduced to levels that prevent or greatly delay asbestosis, as well as others whose exposures are indirect, have a lower risk of lung cancer than those with higher and more direct exposures.

Workers who began employment in a British textile mill after 1933, when implementation of the Asbestos Industry Regulations of 1931 reduced (but did not abolish) dust exposures, were reported in 1968 to show no excess of neoplasms.[82] The long latent periods of asbestos-related lung cancer and mesothelioma, which would probably be even longer at lower dose levels, are such that it is too soon to draw final conclusions as to the eventual incidence of these malignancies. Nevertheless, reduced exposure seems to be having an effect. Another indication of reduced incidence or delayed onset of disease with lower exposure is in the observations of Newhouse,[112] who found that, although there were more deaths from lung cancer and chronic respiratory disease among those who had heavy exposures many years previously in a London asbestos-products plant, this was not true among those who had low or moderate exposures. McDonald et al.[101] recently reported the mortality experience of men who worked in chrysotile mines and mills of Quebec. There was a slight excess of lung cancer among the 2457 deaths in workers born 1890-1920, but all could be explained by the excess that

occurred in those who had been maximally exposed. This suggests that, insofar as chrysotile miners and millers are concerned, the risk drops off rapidly with decreasing accumulated dosage.

Most series of case reports of mesothelioma include some persons who have worked in the construction or shipbuilding industries, but in trades not involving direct contact with asbestos.[102] Such persons as plumbers, electricians, and metal workers often have more ferruginous bodies in their lungs than do white-collar workers.[134] Although Dunn and Weir,[33] in a study of occupational groups in California that revealed an excess of deaths from lung cancer in insulation workers, found no excess lung-cancer deaths in other construction trades, the groups they studied were diluted with many persons who were unlikely to have had exposures. Nevertheless, there may be a definable gradient of effect within the construction trades. More thorough studies of groups with indirect exposures are certainly needed.

We cannot extrapolate from the mortality experience of those who are directly and indirectly exposed to asbestos in their employment to the general public who have had moderate or slight exposures from ambient air. There is evidence to suggest a gradient of effect from direct occupational, to indirect occupational, to family and neighborhood situations, in all of which dust concentrations are probably high by comparison with most community air. This suggests that there are levels of asbestos exposure that will not be associated with any detectable risk. What those levels are is not known, but there is no evidence that persons in the general population--without occupational, household, or neighborhood exposures--have any increased risk of neoplasm, even though there may be ferruginous bodies of fibers in their lungs.

CHAPTER 4

SOURCES OF ASBESTOS FIBERS IN AMBIENT AIR

Precise information is not available on tonnages, numbers of fibers, fiber sizes and varieties, atmospheric dispersion, and ultimate fate of the asbestos emitted into ambient air. Although there are no reliable data to justify extrapolation from the more completely studied occupational exposure experience, information regarding actual and potential sources of emissions of asbestos fibers is of value both for directing future studies and for understanding the steps that might be taken now to safeguard the health of the public.

NATURAL SOURCES OF AIRBORNE ASBESTOS FIBERS

Several varieties of asbestos ore and counterpart rock (containing EM-sized asbestos fibers) occur as outcroppings or are just below the surface of the earth throughout the world. Asbestos fibers can become airborne from these formations during road-building, construction, and tilling of the soil, as well as by landslides, erosion, and weathering. Talc, mined and used extensively in the United States, exists in fibrous, as well as platy, form. Like asbestos ore and rock, talc exists on or close to the earth's surface and is subject to disseminating forces. Such naturally occurring talc, as well as the large quantities used as a diluent and carrier for pesticides, can add to the background fiber concentration in the ambient air.[166,167] It is thought that studies of fibers in glacial and polar ice now under way will permit comparisons of recent deposition with those in the past and thereby provide definite information on the relative contributions of natural and industrial sources.

MINING AND MILLING OF ASBESTOS

Mining and milling of asbestos provides another source of asbestos emissions. In the United States, such activity is presently confined to a few mines in California, Vermont, Arizona, and North Carolina. Fibers are emitted during removal of overburden and preparation of the ore body for open-pit mining. Further release occurs during drilling and ore-breaking. Waste dumps from mining and milling are exposed to wind and to disturbance by bulldozing. Fibers are emitted during drying, crushing, grinding, and screening of the ore. If dust collectors and air-cleaning devices are used, disposal of the collected dust provides a potential source of fiber emission.

TRANSPORTATION OF MATERIALS CONTAINING ASBESTOS

Transportation of asbestos ore, milled asbestos fiber, and asbestos-containing products and wastes is an emission source of varying importance. Movement of asbestos ore from mine to mill in open trucks contributes to the overall emission. The shipment of milled asbestos fiber, usually in bags, can result in emissions. If bags are reused, either in the asbestos industry or elsewhere, they will become a source of fibers. Occasionally, bags are broken and asbestos is spilled during handling. Similar emissions

occur during the shipment of products. Transporting asbestos-containing solid wastes in open vehicles through urban areas can be a more important emission source.

MANUFACTURE OF PRODUCTS CONTAINING ASBESTOS

Industries that must provide ventilation and other dust-control measures for the protection of workers may emit asbestos fibers into the surrounding environment unless effective air cleaning is applied to effluents. Fibers removed by ventilation and filtering devices and not reintroduced into the production process and asbestos-containing waste products of the manufacturing process ultimately are disposed of outside the plant.

USE OF PRODUCTS CONTAINING ASBESTOS

Many products, at times unknown to the user, contain asbestos of one kind or another. There are great variations among such products with respect to the chances of fiber release during the use of the product. The likelihood depends predominantly on the ease with which the fibers can be dislodged by the application of energy and on the degree to which the application of energy actually destroys the fibers during the use of the product. Almost all the asbestos fibers used in the United States for manufacturing products becomes tightly bound within the products and undergoes little actual abrasion or wear before being discarded. Asbestos cement products (accounting for most of the asbestos used in the United States), shingles, and floor tiles are in this category. Some asbestos-containing products, such as brakelinings, are subjected to high energy, and their rate of wear is considerable and at times almost complete. In the case of brakelinings, the application of energy is so intense and the heat created so great that

most chrysotile fibers are destroyed by being converted to another substance, which is nonfibrous.[65,68,89] Nevertheless, an appreciable percentage (1%-3%) remains as fibrous asbestos. In some products--for example, asbestos cloth, paper, and sprayed fireproofing materials--asbestos fibers are not tightly bound or mixed with other material that holds them in place. Fiber release from these products occurs primarily during application and removal. The spray fireproofing of buildings with asbestos-containing materials is a case in point. This operation can be a serious source of emission, in that it usually occurs in densely populated areas. The total amount of asbestos fiber used in such procedures, however, is relatively small.

Talc is mined and milled and used in greater quantities than is asbestos in the United States. Because it contains asbestos fibers, its uses will add to the total number of fibers (including nonasbestos fibers) emitted. The use of talc in dispensing pesticides over wide areas of the country and its use in cosmetics are two examples of how this material may act as a source of asbestos and other fibers.

DEMOLITION

For years, asbestos has been incorporated in building materials. In some forms of insulation and wallboard, the amount present is less than 20% of the total; but other materials consist mostly or entirely of asbestos. When a building is demolished, areas of loosened asbestos are open to the ambient air and fibers are emitted. In general, single-family residential structures contain only small amounts of asbestos insulation. Demolition of industrial and commercial buildings that have been fireproofed

with asbestos-containing materials will prove to be an emission source in the future, requiring control measures.

SOLID-WASTE DISPOSAL

Solid wastes produced during manufacture of asbestos-containing products, use of such products, and demolition can be emission sources. These waste materials are usually disposed of without regard to their potential as emission sources. Alternate methods of disposal often result in commingling of asbestos-containing wastes with municipal wastes in open dumps and thus create a long-term emission source.

CHAPTER 5

PRINCIPLES OF CONTROL

The natural background level of asbestos fibers is difficult to quantify or modify. The major sources of local ambient-air contamination with asbestos are industrial processing and use of products containing asbestos. It is feasible to identify the sources of emission, select those to which presently available control procedures can be applied, and point out areas that need further study and development of new methods of control.

NATURAL SOURCES OF AIRBORNE ASBESTOS FIBERS

Natural sources of asbestos fibers have been identified in many areas of the United States. It is assumed that emissions of fibers from these reservoirs through erosion and wind make up a natural background of asbestos in ambient air. Few data are available on the magnitude of the contamination from natural sources, variation with geographic location, and seasonal variation. It is desirable to have information on the natural background, because it would assist in evaluating the effects of control measures and permit some estimates of the lower limits of contamination that might be achieved in different areas of the country.

MINING OF ASBESTOS

The standard techniques for dust control in underground or open-pit mines, if not already in use, can be applied in asbestos mines. Roadways in open pits should be treated with dust-suppressive agents; truckloads of ore should be covered with tarpaulins while being transported; handling of ore should be minimized; ore storage piles should be moistened to reduce wind erosion; and waste dumps should be treated with dust-suppressive agents.

MILLING OF ASBESTOS

An in-plant dust-control program for protecting workers in asbestos mills includes exhaust ventilation with efficient dust-collecting and air-cleaning equipment, isolation, enclosure, wet methods, and good housekeeping and maintenance.

MANUFACTURE OF PRODUCTS CONTAINING ASBESTOS

The elements of dust control recommended for the milling of asbestos ore also apply to the manufacture of asbestos-containing products. It is important that a dust-control system be specific to the operation for which it is used and that it be tested to ensure its effectiveness. Only persons especially trained and experienced in dust control should be used to develop and institute dust-control procedures.

USE OF PRODUCTS CONTAINING ASBESTOS

An important emission source is the use of insulating materials containing asbestos. This constitutes only a small fraction of the asbestos used. But the asbestos in some insulating materials is not bound as it is in cementitious products or tiles; construction activities usually are carried on in urban areas where many people are exposed to contaminated air; and control of dust during construction, although feasible, is difficult.

The most effective approach in reducing exposures of the general public to asbestos from this source involves controlling dust production and release at its origin during construction work. The generation of dust should be reduced by changing material-handling methods, work practices, and cleanup procedures. Local exhaust systems should be used for dust collection at points of generation; for example, dust collectors for band

saws are available, and hand-powered tools supplied with exhaust systems are being made. Much developmental work is needed to produce portable air-moving and air-cleaning systems for use in tunnels, crawl spaces, and other confined spaces. This subject has been neglected by industrial hygienists and ventilation engineers, and no satisfactory equipment is available.

DEMOLITION AND WASTE DISPOSAL

Demolition and waste disposal are likely to be emission sources if appreciable amounts of asbestos are used in construction, unless operational procedures are strictly controlled. Isolation, enclosure, and wetting down are useful. Caution must be observed not to demolish during high winds and to keep sludge from drying out and becoming airborne later through natural forces and from being introduced into sources of drinking water.

MEASUREMENT OF AIRBORNE ASBESTOS

An important consideration in the development of a strategy for control is whether there are methods for measuring airborne concentrations of asbestos that are sufficiently sensitive, specific, and reproducible. Present methods of sampling, identifying, and measuring airborne asbestos are not entirely satisfactory, especially if one is dealing with low concentrations and unidentified or mixed sources. Only within recent years have methods for determining concentrations of fibers for industrial hygiene purposes been standardized;[14,35] they use samples collected on membrane filters in which fibers are counted with phase-contrast illumination. Electron microscopic methods give a much more complete indication of the total fiber content

of the air; but when the need for fiber identification is included, they are tedious and expensive for routine use.[1,66,137] The relative biologic significance of different sizes of fibers is not known, nor is the relative importance of fiber numbers and fiber mass. There appear to be no published data on the efficiency of air-cleaning equipment as related to fibers of different sizes.

In spite of the difficulties, it is possible to sample air, determine the approximate concentration of airborne fibers, and identify the major types of asbestos. It is not desirable to limit environmental measurements to a single method until there is a clearer definition of the critical variables in terms of health. Because of methodologic and other uncertainties, it is not yet feasible to base control on numerical ambient air quality standards.

CHAPTER 6

RESEARCH NEEDS

Two recent reports[25,167] have discussed in some detail the many kinds of research needed to answer pressing questions concerning the effects of asbestos on health and the degree and nature of necessary controls. Investigations along the following lines should be given high priority.

Study of the mechanism of action of the asbestos minerals should continue, with particular attention to carcinogenic effects. It is important to learn more about the influence of asbestos type and fiber size on respirability, deposition, retention, translocation, and effects at the tissue, cellular, and molecular levels, with and without cofactors. It is especially important that the role of fibers below the LM range be clarified.

Methods of sampling, identifying, and quantitating airborne asbestos need continued development. Coordination with studies in animals and man is essential to ensure that environmental data will be biologically relevant. Similarly, methods for identifying and quantitating asbestos in biologic tissue need development and application.

Quantitative methods for measuring airborne asbestos should be applied widely to determine the natural background and the concentration and distribution of fibers in the air near various sources. Conventional LM methods and EM methods should be applied simultaneously in selected occupational and community situations.

More epidemiologic studies are needed. Populations in several different exposure ranges should be studied, including occupational, household, and neighborhood exposures. Special studies of mesothelioma are needed to determine whether the incidence has been increasing and to determine the current pattern of distribution. A large series of routine autopsies should be studied to determine whether causes of death can be related to amounts of asbestos in the lungs and other organs. All the above are urgent if a range of safe exposure is to be established with confidence.

CHAPTER 7

CONCLUSIONS AND RECOMMENDATIONS

PATHOGENICITY OF ASBESTOS MINERALS

Any of the commercially used asbestos minerals, when inhaled in sufficient numbers, as in uncontrolled occupational exposures, can cause disabling fibrosis of the lungs. An association between occupational exposures to asbestos and bronchogenic carcinoma has been established, but the dose relationship and the role of cofactors have not been defined. Evidence of a causal association between some but not all exposures to asbestos fibers and diffuse malignant mesotheliomas of the pleura and peritoneum is substantial, but evidence of such a relationship with other tumors is inconclusive. Although the different types of asbestos differ in some of their biologic effects, no type can be regarded as free of hazard. The hypothesis that asbestos fibers act as cofactors or carriers of carcinogens is attractive, but as yet unproved.

EVIDENCE OF HUMAN NONOCCUPATIONAL EXPOSURES TO ASBESTOS

The demonstration of ferruginous bodies, similar to those found in asbestos workers, in a large proportion of randomly selected lung specimens in many parts of the world is presumptive evidence that persons with no occupational contact may have inhaled and retained asbestos. Proof has come in some areas with positive identification of chrysotile asbestos fibers. Analyses of community air for asbestos have been too limited to define the sources, concentrations, and distribution of fibers in the environment. The fiber concentrations that have been demonstrated in ambient air are small, compared with those in industry, but data are inadequate for definitive comparisons.

ESTIMATION OF RISK IN HUMAN NONOCCUPATIONAL EXPOSURES TO ASBESTOS

The most important question in the case of persons with nonoccupational exposures to asbestos is whether there is an increased risk of malignancies. Industrial experience indicates that there is no likelihood of significant asbestosis in nonoccupational exposures. The major potential for risk appears to lie in those with indirect occupational contacts, household contacts, or residence in the immediate neighborhood of asbestos sources; and even there, the actual risk is poorly defined. But the fact that there appears to be a gradient of effect in such groups suggests that there are levels of inhaled asbestos without detectable risk. It is not known what range of respirable airborne asbestos fibers will ultimately be found to have no measurable effects on health. At present, there is no evidence that the small numbers of fibers found in most members of the general population affect health or longevity.

NEED FOR AND FEASIBILITY OF CONTROLS

Asbestos is too important in our technology and economy for its essential use to be stopped. But, because of the known serious effects of uncontrolled inhalation of asbestos minerals in industry and uncertainty as to the shape and character of the dose-response curve in man, it would be highly imprudent to permit unrestricted additional contamination of the public environment with asbestos. Continued use at minimal risk to the public requires that the major sources of man-made asbestos emission into the atmosphere be defined and controlled. In the absence of such controls, local fiber concentrations might at times approach those in occupational sites. Analytic methods and epidemiologic data are inadequate for the development of ambient air standards, but emission controls are needed and appear feasible.

REFERENCES

1. Alcocer, A. E., J. Murchio, and P. K. Mueller. Asbestos Content of Some Urban Air Samples. AIHL Report No. 90 (revised). Berkeley: State of California Department of Public Health, 1970. 12 pp.

2. American Conference of Governmental Industrial Hygienists. Threshold Limit Values of Airborne Contaminants and Intended Changes Accepted by ACGIH for 1970. Cincinnati: American Conference of Governmental Industrial Hygienists, 1970. 27 pp.

3. Anjilvel, L., and W. M. Thurlbeck. The incidence of asbestos bodies in the lungs at random necropsies in Montreal. Canad. Med. Ass. J. 95:1179-1182, 1966.

4. Anspach, M. Sind Pleuraverkalkungen pathognomonisch für eine Asbestose? Int. Arch. Gewerbepath. u. Gewerbehyg. 19:108-120, 1962.

5. Anton, H. C. Multiple pleural plaques. Brit. J. Radiol. 40:685-690, 1967.

6. Anton, H. C. Multiple pleural plaques: Part II. Brit. J. Radiol. 41:341-348, 1969.

7. Asbestos Industry Regulations, 1931. Statutory Rules and Orders, 1931, No. 1140. London: H. M. Stationery Office, 1931. 4 pp.

8. Ashcroft, T. Asbestos bodies in routine necropsies on Tyneside: a pathological and social study. Brit. Med. J. 1:614-618, 1968.

9. Ashcroft, T., and A. G. Heppleston. Mesothelioma and asbestos on Tyneside. A pathological and social study. Presented at International Conference on Pneumoconiosis, Johannesburg, April 23-May 2, 1969.

10. Balzer, J. L., W. C. Cooper, and D. F. Fowler. Fibrous lined air transmission systems: an assessment of their environmental effects. Arch. Environ. Health (in press)

11. Bonser, G. M., J. S. Faulds, and M. J. Stewart. Occupational cancer of the urinary bladder in dyestuffs operatives and of the lung in asbestos textile workers and iron-ore miners. Amer. J. Clin. Path. 25:126-134, 1955.

12. Borow, M., A. Conston, L. L. Livornese, and N. Schalet. Mesothelioma and its association with asbestosis. J.A.M.A. 201:587-591, 1967.

13. Braun, D. C., and T. D. Truan. An epidemiological study of lung cancer in asbestos miners. A.M.A. Arch. Industr. Health 17:634-653. 1958.

14. British Occupational Hygiene Society. Hygiene standards for chrysotile asbestos dust. Ann. Occup. Hyg. 11:47-69, 1968.

15. Buchanan, W. D. Asbestosis and primary intrathoracic neoplasms. Ann. N. Y. Acad. Sci. 132:507-518, 1965.

16. Byrom, J. C., A. A. Hodgson, and S. Holmes. A dust survey carried out in buildings incorporating asbestos-based material in their construction. Ann. Occup. Hyg. 12:141-145, 1969.

17. Cauna, D., R. S. Totten, and P. Gross. Asbestos bodies in human lungs at autopsy. J.A.M.A. 192:371-373, 1965.

18. Churg, J., S. H. Rosen, and S. Moolten. Histological characteristics of mesothelioma associated with asbestos. Ann. N. Y. Acad. Sci. 132:614-622, 1965.

19. Cooke, W. E. Asbestos dust and the curious bodies found in asbestosis. Brit. Med. J. 2:578-580, 1929.

20. Cooke, W. E. Fibrosis of the lungs due to the inhalation of asbestos dust. Brit. Med. J. 2:147, 1924.

21. Cooke, W. E. Pulmonary asbestosis. Brit. Med. J. 2:1024-1025, 1927.

22. Cooper, W. C. Asbestos as a hazard to health. Arch. Environ. Health 15:285-290, 1967.

23. Cooper, W. C., and J. L. Balzer. Evaluation and control of asbestos exposures in the insulating trade. Presented at the 2nd International Conference on Biological Effects of Asbestos, Dresden, April 22-25, 1968.

24. Cordova, J. F., H. Tesluk, and K. P. Knudtson. Asbestosis and carcinoma of the lung. Cancer 15:1181-1187, 1962.

25. Cralley, L. J., W. C. Cooper, W. S. Lainhart, and M. C. Brown. Research on health effects of asbestos. J. Occup. Med. 10:38-41, 1968.

26. Cralley, L. J., R. G. Keenan, R. E. Kupel, R. E. Kinser, and J. R. Lynch. Characterization and solubility of metals associated with asbestos fibers. Amer. Industr. Hyg. Ass. J. 29:569-573, 1968.

27. Cralley, L. J., R. G. Keenan, and J. R. Lynch. Exposure to metals in the manufacture of asbestos textile products. Amer. Industr. Hyg. Ass. J. 28:452-461, 1967.

28. Cralley, L. J., R. G. Keenan, J. R. Lynch, and W. S. Lainhart. Source and identification of respirable fibers. Amer. Industr. Hyg. Ass. J. 29:129-135, 1968.

29. Davis, J. M. G. Electron-microscope studies of asbestosis in man and animals. Ann. N. Y. Acad. Sci. 132:98-111, 1965.

30. Dicke, T. E., and B. Naylor. Prevalence of "asbestos" bodies in human lungs at necropsy. Dis. Chest 56:122-125, 1969.

31. Doll, R. Mortality from lung cancer in asbestos workers. Brit. J. Industr. Med. 12:81-86, 1955.

32. Dreessen, W. C., J. M. Dallavalle, T. I. Edwards, J. W. Miller, and R. R. Sayers. A Study of Asbestos in the Asbestos Textile Industry. (Public Health Bulletin No. 241) Washington: U. S. Government Printing Office, 1938. 126 pp.

33. Dunn, J. E., Jr., and J. M. Weir. A prospective study of mortality of several occupational groups. Special emphasis on lung cancer. Arch. Environ. Health 17:71-76, 1968.

34. Dunn, J. E., Jr., and J. M. Weir. Cancer experience of several occupational groups followed prospectively. Amer. J. Public Health 55:1367-1375, 1968.

35. Edwards, G. H., and J. R. Lynch. The method used by the U. S. Public Health Service for enumeration of asbestos dust on membrane filters. Ann. Occup. Hyg. 11:1-6, 1968.

36. Elmes, P. C., W. T. E. McCaughey, and O. L. Wade. Diffuse mesothelioma of the pleura and asbestos. Brit. Med. J. 1:350-353, 1965.

37. Elmes, P. C., and O. L. Wade. Relationship between exposure to asbestos and pleural malignancy in Belfast. Ann. N. Y. Acad. Sci. 132:549-557, 1965.

38. Elwood, P. C., and A. L. Cochrane. A follow-up study of workers from an asbestos factory. Brit. J. Industr. Med. 21:304-307, 1964.

39. Enterline, P. E. Mortality among asbestos products workers in the United States. Ann. N. Y. Acad. Sci. 132:156-165, 1965.

40. Enterline, P. E., and M. A. Kendrick. Asbestos-dust exposures at various levels and mortality. Arch. Environ. Health 15:181-186, 1967.

41. Enticknap, J. B., and W. J. Smither. Peritoneal tumours in asbestosis. Brit. J. Industr. Med. 21:20-31, 1964.

42. Fowler, P. B. S., J. C. Sloper, and E. C. Warner. Exposure to asbestos and mesothelioma of the pleura. Brit. Med. J. 2:211-213, 1964.

43. Frost, J., J. Georg, and P. Flemming Møller. Asbestosis with pleural calcification among insulation workers. Danish Med. Bull. 3:202-204, 1956.

44. Gardner, L. U. Chrysotile asbestos as an indicate of subtile differences in animal tissues. Amer. Rev. Tuberc. 45:762-766, 1941.

45. Gardner, L. U., and D. E. Cummings. Studies on experimental pneumokoniosis. VI. Inhalation of asbestos dust: its effect upon primary tuberculous infection. J. Industr. Hyg. 13:65-81, 97-114, 1931.

46. Gaze, R. The physical and molecular structure of asbestos. Ann. N. Y. Acad. Sci. 132:23-30, 1965.

47. Ghezzi, I., G. Molteni, and U. Puccetti. Asbestos bodies in the lungs of inhabitants of Milan. Med. Lavoro 58:223-227, 1967.

48. Gibbs, G. W. Some problems associated with the storage of asbestos in polyethylene bags. Amer. Industr. Hyg. Ass. J. 30:458-464, 1969.

49. Gilson, J. C. Wyers memorial lecture, 1965. Health hazards of asbestos. Recent studies on its biological effects. Trans. Soc. Occup. Med. 16:62-74, 1966.

50. Gloyne, S. R. Pneumoconiosis. A histological survey of necropsy material in 1205 cases. Lancet 1:810-814, 1951.

51. Gough, J. Differential diagnosis in the pathology of asbestosis. Ann. N. Y. Acad. Sci. 132:368-372, 1965.

52. Graham, J. and R. Graham. Ovarian cancer and asbestos. Environ. Res. 1:115-128, 1967.

53. Gross, P. Today's pressing question. How safe is urban ambient air? (Letter to the editor.) Arch. Path. 82:195, 1966.

54. Gross, P., L. J. Cralley, and R. T. P. deTreville. "Asbestos" bodies: their nonspecificity. Amer. Industr. Hyg. Ass. J. 28:541-542, 1967.

55. Gross, P., and R. T. P. deTreville. Experimental asbestosis. Studies on the progressiveness of the pulmonary fibrosis caused by chrysotile dust. Arch. Environ. Health 15:638-649, 1967.

56. Gross, P., R. T. P. deTreville, and L. J. Cralley. Pulmonary ferruginous bodies. Development in response to filamentous dusts and a method of isolation and concentration . Arch. Path. 85:539-546, 1968.

57. Gross, P., R. T. P. deTreville, and M. N. Haller. Asbestos versus nonasbestos fibers. Ultra microscopic criteria. Arch. Environ. Health 20:571-578, 1970.

58. Gross, P., R. T. P. deTreville, E. B. Tolker, M. Kaschak, and M. A. Babyak. Experimental asbestosis. The development of lung cancer in rats with pulmonary deposits of chrysotile asbestos dust. Arch. Environ. Health 15:343-355, 1967.

59. Hammond, E. C., I. J. Selikoff, and J. Churg. Neoplasia among insulation workers in the United States with special reference to intra-abdominal neoplasia. Ann. N. Y. Acad. Sci. 132:519-525, 1965.

60. Hardy, H. L. Asbestos related disease. Amer. J. Med. Sci. 250:381-389, 1965.

61. Harington, J. S. Chemical studies of asbestos. Ann. N. Y. Acad. Sci. 132:31-47, 1965.

62. Harington, J. S. Occurrence of oils containing 3:4 benzpyrene and related substances in asbestos. Nature 193:43-45, 1962.

63. Harington, J. S., and F. J. C. Roe. Studies of carcinogenesis of asbestos fibers and their natural oils. Ann. N. Y. Acad. Sci. 132:439-450, 1965.

64. Harington, J. S., and M. Smith. Studies of hydrocarbons on mineral dusts. The elution of 3:4-benzpyrene and oils from asbestos and coal dusts by steam. Arch. Environ. Health 8:453-458, 1964.

65. Hatch, D. Possible alternatives to asbestos as a friction material. Ann. Occup. Hyg. 13:25-29, 1970.

66. Heffelfinger, R. E., C. W. Melton, and W. M. Henry. Interim Report on Development of a Rapid Survey Method of Sampling and Analysis for Asbestos in Ambient Air to National Center for Air Pollution Control. Columbus: Battelle Memorial Institute, 1970. 24 pp.

67. Hendry, N. W. The geology, occurrences, and major uses of asbestos. Ann. N. Y. Acad. Sci. 132:12-22, 1965.

68. Hickish, D. E., and K. L. Knight. Exposure to asbestos during brake maintenance. Ann. Occup. Hyg. 13:17-21, 1970.

69. Holt, P. F., J. Mills, and D. K. Young. Experimental asbestosis in the guinea-pig. J. Path. Bact. 92:185-195, 1966.

70. Hourihane, D. O'B. The pathology of mesotheliomata and an analysis of their association with asbestos exposure. Thorax 19:268-278, 1964.

71. Hourihane, D. O'B., L. Lessof, and P. C. Richardson. Hyaline and calcified pleural plaques as an index of exposure to asbestos. A study of radiological and pathological features of 100 cases with a consideration of epidemiology. Brit. Med. J. 1:1069-1074, 1966.

72. Hromek, J. Large scale incidence of characteristic pleural changes in the inhabitants of the western section of the former Jihlava region. Rozhl. Tuberk. 22:405-418, 1962. (in Czech)

73. International Agency for Research on Cancer. Annual Report, 1969, p. 34. Lyon: World Health Organization, 1970.

74. Isselbacher, K. J., H. Klaus, and H. L. Hardy. Asbestosis and bronchogenic carcinoma. Report of one autopsied case and review of the available literature. Amer. J. Med. 15:721-732, 1953.

75. Jacob, G., and M. Anspach. Pulmonary neoplasia among Dresden asbestos workers. Ann. N. Y. Acad. Sci. 132:536-548, 1965.

76. Jacob, G., and H. Bohlig. Uber Haufigkeit und Besonderheiten des Lungenkrebses bei Asbestose. Arch. Gewerbepath. u. Gewerbehyg. 14:10-28, 1955.

77. Keal, E. E. Asbestosis and abdominal neoplasms. Lancet 2:1211-1216, 1960.

78. Keenan, R. G., and J. R. Lynch. Techniques for the detection, identification and analysis of fibers. Amer. Industr. Hyg. Ass. J. 31:587-597, 1970.

79. Kiviluoto, R. Pleural calcification as a roentgenologic sign of non-occupational endemic anthophyllite-asbestosis. Acta. Radiol. Suppl. 194:1-67, 1960.

80. Kleinfeld, M., J. Messite, and O. Kooyman. Mortality experience in a group of asbestos workers. Arch. Environ. Health 15:177-180, 1967.

81. Knox, J. F., R. S. Doll, and I. D. Hill. Cohort analysis of changes in incidence of bronchial carcinoma in a textile asbestos factory. Ann. N. Y. Acad. Sci. 132:526-535, 1965.

82. Knox, J. F., S. Holmes, R. Doll, and I. D. Hill. Mortality from lung cancer and other causes among workers in an asbestos textile factory. Brit. J. Industr. Med. 25:293-303, 1968.

83. Laamanen, A., L. Noro, and V. Raunio. Observations on atmospheric air pollution caused by asbestos. Ann. N. Y. Acad. Sci. 132:240-245, 1965.

84. Langer, A. M., V. Baden, E. C. Hammond, and I. J. Selikoff. Inorganic fibers, including chrysotile, in lungs at autopsy. Preliminary report. Presented at Third International Symposium on Inhaled Particles, London, September 22, 1970.

85. Langer, A. M., I. J. Selikoff, and A. Sastre. Chrysotile asbestos in the lungs of persons in New York City. Arch. Environ. Health 22:348-361, 1971.

86. Leicher, F. Primärer Deckzellentumor des Bauchfells bei Asbestose. Arch. Gewerbepath. u. Gewerbehyg. 13:382-392, 1954.

87. Lieben, J. Malignancies in asbestos workers. Arch. Environ. Health 13:619-621, 1966.

88. Lieben, J., and H. Pistawka. Mesothelioma and asbestos exposure. Arch. Environ. Health 14:559-563, 1967.

89. Lynch, J. R. Brake lining decomposition products. J. Air Poll. Control Ass. 18:824-826, 1968.

90. Lynch, J. R., H. E. Ayer, and D. L. Johnson. The interrelationships of selected asbestos exposure indices. Amer. Industr. Hyg. J. 31:598-604, 1970.

91. Lynch, K. M., and W. A. Smith. Pulmonary asbestosis III: Carcinoma of lung in asbesto-silicosis. Amer. J. Cancer 24:56-64, 1935.

92. MacNab, G., and J. S. Harington. Haemolytic activity of asbestos and other mineral dusts. Nature 214:522-523, 1967.

93. Mancuso, T. F., and E. J. Coulter. Methodology in industrial health studies. The cohort approach, with special reference to an asbestos company. Arch. Environ. Health 6:210-226, 1963.

94. Mancuso, T. F., and A. A. El-Attar. Mortality pattern in a cohort of asbestos workers. J. Occup. Med. 9:147-162, 1967.

95. Mann, R. H., J. L. Grosh, and W. M. O'Donnell. Mesothelioma associated with asbestosis. Cancer 19:521-526, 1966.

96. Marchand, F. Ueber eigentumliche Pigmentkristalle in der Lungen. Verh. Deutsche Ges. Path. 17:223-228, 1907.

97. Marsová, D. Beitrag zur Ätiologie der Pleuraverkalkungen. Z. Tuberk. 121:329-334, 1964.

98. McCaughey, W. T. E. Criteria for diagnosis of diffuse mesothelial tumors. Ann. N. Y. Acad. Sci. 132:603-613, 1965.

99. McCaughey, W. T. E., O. L. Wade, and P. C. Elmes. Exposure to asbestos dust and diffuse pleural mesotheliomas. Brit. Med. J. 2:1397, 1962.

100. McDonald, A. D., A. Harper, O. A. El Attar, and J. C. McDonald. Epidemiology of primary malignant mesothelial tumors in Canada. Cancer 26:914-919, 1970.

101. McDonald, J. C., A. D. McDonald, G. W. Gibbs, J. Siemiatycki, and C. E. Rossiter. Mortality from lung cancer and other causes in the chrysotile asbestos mines and mills of Quebec. Arch. Environ. Health. (in press)

102. McEwen, J., A. Finlayson, A. Mair, and A. A. M. Gibson. Mesothelioma in Scotland. Brit. Med. J. 4:575-578, 1970.

103. McVittie, J. C. Asbestosis in Great Britain. Ann. N. Y. Acad. Sci. 132:128-138, 1965.

104. Merewether, E. R. A. Asbestosis and carcinoma of the lung, in Annual Report of the Chief Inspector of Factories for the Year 1947. London: H. M. Stationery Office, 1949. 79 pp.

105. Merewether, E. R. A. The occurrence of pulmonary fibrosis and other pulmonary affections in asbestos workers. J. Industr. Hyg. 12:198-222, 239-257, 1930.

106. Merewether, E. R. A., and C. W. Price. Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry. London: His Majesty's Stationery Office, 1930. 34 pp.

107. Meurman, L. O. Asbestos bodies and pleural plaques in a Finnish series of autopsy cases. Acta Path. Microbiol. Scand. Suppl. 181:1-107, 1966.

108. Meurman, L. O. Pleural fibrocalcific plaques and asbestos exposure. Environ. Res. 2:30-46, 1968.

109. Miller, L., W. E. Smith, and S. W. Berliner. Tests for effect of asbestos on benzo(a)pyrene carcinogenesis in the respiratory tract. Ann. N. Y. Acad. Sci. 132:489-500, 1965.

110. Mills, R. G. Pulmonary asbestosis: report of a case. Minn. Med. 13:495-499, 1930.

111. Murray, H. M. Statement before the committee in the minutes of evidence, pp. 127-128. In Report of the Departmental Committee on Compensation for Industrial Disease. London: His Majesty's Stationery Office, 1907.

112. Newhouse, M. L. A study of the mortality of workers in an asbestos factory. Brit. J. Industr. Med. 26:294-301, 1969.

113. Newhouse, M. L., and H. Thompson. Epidemiology of mesothelial tumors in the London area. Ann. N. Y. Acad. Sci. 132:579-588, 1965.

114. Newhouse, M. L., and H. Thompson. Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. Brit. J. Industr. Med. 22:261-269, 1965.

115. Owen, W. G. Diffuse mesothelioma and exposure to asbestos dust in the Merseyside area. Brit. Med. J. 2:214-218, 1964.

116. Owen, W. G. Mesothelial tumors and exposure to asbestos dust. Ann. N. Y. Acad. Sci. 132:674-679, 1965.

117. Parazzi, E., B. Pernis, G. C. Secchi, and E. C. Vigliani. Studies on in vitro cytotoxicity of asbestos dust. Med. Lavoro 59:561-576, 1968.

118. Polliack, A., and M. I. Sacke. Prevalence of asbestos bodies in basal lung smears. Israel J. Med. Sci. 4:223-226, 1968.

119. Pooley, F. D., P. D. Oldham, C.-H. Um, and J. C. Wagner. The detection of asbestos in tissues. Presented at International Conference on Pneumoconiosis, Johannesburg, April 23-May 2, 1969.

120. Raunio, V. Occurrence of unusual pleural calcification in Finland. Studies on atmospheric pollution caused by asbestos. Ann. Med. Int. Fenn. 55:1-61, (suppl. 49), 1966.

121. Readling, C. L. Asbestos. In: U. S. Department of Interior. Bureau of Mines Mineral Yearbook. Washington: U. S. Government Printing Office, 1969. 9 pp. (preprint)

122. Roach, S. A. Measurement of airborne asbestos dust by instruments measuring different parameters. Ann. N. Y. Acad. Sci. 132:306-315, 1965.

123. Roberts, G. H. Asbestos bodies in lungs at necropsy. J. Clin. Path. 20:570-573, 1967.

124. Roe, F. J. C., R. L. Carter, M. A. Walters, and J. S. Harington. The pathological effects of subcutaneous injections of asbestos fibres in mice: migration of fibres to submesothelial tissues and induction of mesotheliomata. Int. J. Cancer 2:628-638, 1967.

125. Rosato, D. V. Asbestos. Its Industrial Applications. New York: Reinhold Publ. Corp., 1959. 214 pp.

126. Rous, V., and J. Studený. Aetiology of pleural plaques. Thorax 25:270-284, 1970.

127. Schnitzer, R. J., and Pundsack, F. L. Asbestos hemolysis. Environ. Res. 3:1-13, 1970.

128. Schuster, N. H. Pulmonary asbestosis in a dog. J. Path. Bact. 34:751-757, 1931.

129. Secchi, G. C., and A. Rezzonico. Hemolytic activity of asbestos dust. Med. Lavoro 59:1-5, 1968.

130. Selikoff, I. J. The occurrence of pleural calcification among asbestos insulation workers. Ann. N. Y. Acad. Sci. 132:351-367, 1965.

131. Selikoff, I. J., J. Churg, and E. C. Hammond. Asbestos exposure and neoplasia. J.A.M.A. 188:22-26, 1964.

132. Selikoff, I. J., J. Churg, and E. C. Hammond. Relation between exposure to asbestos and mesothelioma. New Eng. J. Med. 272:560-565, 1965.

133. Selikoff, I. J., and E. C. Hammond. Asbestos bodies in the New York City population in two periods of time. Presented at International Conference on Pneumoconiosis, Johannesburg, April 23-May 2, 1969.

134. Selikoff, I. J., and E. C. Hammond. Environmental epidemiology. III. Community effects of nonoccupational environmental asbestos exposure. Amer. J. Public Health 59:1658-1666, 1968.

135. Selikoff, I. J., E. C. Hammond, and J. Churg. Asbestos exposure, smoking, and neoplasia. J.A.M.A. 204:106-112, 1968.

136. Selikoff, I. J., E. C. Hammond, and J. Churg. Mortality experiences of asbestos installation workers, 1943-1968. Presented at International Conference on Pneumoconiosis, Johannesburg, April 23-May 2, 1969.

137. Selikoff, I. J., W. J. Nicholson, and D. M. Langer. Asbestos air pollution in urban areas. Presented at the American Medical Association Air Pollution Medical Research Conference, New Orleans, October 5, 1970.

138. Sluis-Cremer, G. K. Asbestosis in South African asbestos miners. Environ. Res. 3:310-319, 1970.

139. Smith, W. E., L. Miller, R. E. Elsasser, and D. D. Hubert. Tests for carcinogenicity of asbestos. Ann. N. Y. Acad. Sci. 132:456-488, 1965.

140. Smith, W. E., and E. Yazdi. Induction of carcinomas from mouse lung transplanted with asbestos. Proc. Amer. Ass. Cancer Res. 10:84, 1969. (abstract)

141. Soper, W. B. Pulmonary asbestosis. A report of a case and a review. Amer. Rev. Tuberc. 22:571-584, 1930.

142. Speil, S., and J. P. Leineweber. Asbestos minerals in modern technology. Environ. Res. 2:166-208, 1969.

143. Stanton, M. F., R. Blackwell, and E. Miller. Experimental pulmonary carcinogenesis with asbestos. Amer. Industr. Hyg. Ass. J. 30:236-244, 1969.

144. Stewart, M. J., and A. C. Haddow. Demonstration of the peculiar bodies of pulmonary asbestosis ("asbestosis bodies") in material obtained by lung puncture and in the sputum. J. Path. Bact. 32:172, 1929.

145. Stumphius, J., and P. B. Meyer. Asbestos bodies and mesothelioma. Ann. Occup. Hyg. 11:283-293, 1968.

146. Sullivan, R. J., and Y. C. Athanassidas. Preliminary Air Pollution Survey of Asbestos. A Literature Review. NAPCA Publication No. APTD 69-27. Raleigh: National Air Pollution Central Administration, October 1969.

147. Suzuki, Y., and J. Churg. Structure and development of the asbestos body. Amer. J. Path. 55:75-107, 1969.

148. Tabershaw, I. R. Asbestos as an environmental hazard. J. Occup. Med. 10:32-37, 1968.

149. Thomson, J. G. The pathological diagnosis of malignant mesothelioma. Presented at International Conference on Pneumoconiosis, Johannesburg, April 23-May 2, 1969.

150. Thomson, J. G., and W. M. Graves, Jr. Asbestos as an urban air contaminant. Arch. Path. 81:458-464, 1966.

151. Thomson, J. G., R. O. C. Kaschula, and R. R. MacDonald. Asbestos as a modern urban hazard. S. Afr. Med. J. 37:77-81, 1963.

152. Timbrell, V. The inhalation of fibrous dusts. Ann. N. Y. Acad. Sci. 132:255, 273, 1965.

153. Utidjian, M. D., P. Gross, and R. T. P. deTreville. Ferruginous bodies in human lungs. Prevalence at random autopsies. Arch. Environ. Health 17:327-333, 1968.

154. Vigliani, E. C., I. Ghezzi, P. Maranzana, and B. Pernis. Epidemiological study of asbestos workers in northern Italy. Med. Lavoro 59:481-485, 1968.

155. Vigliani, E. C., G. Mottura, and P. Maranzana. Association of pulmonary tumors with asbestosis in Piedmont and Lombardy. Ann. N. Y. Acad. Sci. 132:558-574, 1965.

156. Vorwald, A. J., T. M. Durkan, and P. C. Pratt. Experimental studies of asbestosis. A.M.A. Arch. Industr. Hyg. Occup. Med. 3:1-43, 1951.

157. Wagner, J. C. Asbestosis in experimental animals. Brit. J. Industr. Med. 20:1-12, 1963.

158. Wagner, J. C. Epidemiology of diffuse mesothelial tumors: Evidence of an association from studies in South Africa and the United Kingdom. Ann. N. Y. Acad. Sci. 132:575-578, 1965.

159. Wagner, J. C. Experimental production of mesothelial tumours of the pleura by implantation of dusts in laboratory animals. Nature 196:180-181, 1962.

160. Wagner, J. C., and G. Berry. Mesotheliomas in rats following inoculation with asbestos. Brit. J. Cancer 23:567-581, 1969.

161. Wagner, J. C., C. A. Sleggs, and P. Marchand. Diffuse pleural mesothelioma and asbestos exposure in the north western Cape Province. Brit. J. Industr. Med. 17:260-271, 1960.

162. Webster, I. Asbestos exposure in South Africa. Presented at International Conference on Pneumoconiosis, Johannesburg, April 23-May 2, 1969.

163. Webster, I. Asbestosis in non-experimental animals in South Africa. Nature 197:506, 1963.

164. Wedler, H.-W. Asbestose und Lungenkrebs. Deutsch. Med. Wschr. 69:575-576, 1943.

165. Weiss, A. Pleurakrebs bei Lungenasbestose, in vivo morphologisch gesichert. Medizinische 3:93-94, 1953.

166. Windom, H., J. Griffin, and E. D. Goldberg. Talc in atmospheric dusts. Environ. Sci. Technol. 1:923-926, 1967.

167. Working Group on Asbestos and Cancer. Report and recommendations of the working group convened under the auspices of the Geographical Pathology Committee of the International Union Against Cancer. Arch. Environ. Health 11:221-229, 1965.

168. Wright, G. W. Asbestos and health in 1969. Amer. Rev. Resp. Dis. 100:467-469, 1969.

169. Wyers, H. Asbestosis. Postgrad. Med. J. 25:631-638, 1949.

170. Zolov, C., T. Bourilkov, and L. Babadjov. Pleural asbestosis in agricultural workers. Environ. Res. 1:287-292, 1967.